SANTA CRUZ BIOTECHNOLOGY, INC.

HXK I (4D7): sc-80978

BACKGROUND

The hexokinases utilize Mg-ATP as a phosphoryl donor to catalyze the first step of intracellular glucose metabolism, the conversion of glucose to glucose-6-phosphate. Four hexokinase isoenzymes have been identified, including hexokinase I (HXK I), hexokinase II (HXK II), hexokinase III (HXK III) and hexokinase IV (HXK IV, also designated glucokinase or GCK). Hexokinases I-III each contain an N-terminal cluster of hydrophobic amino acids. Glucokinase lacks the N-terminal hydrophobic cluster. The hydrophobic cluster is thought to be necessary for membrane binding. This is substantiated by the finding that glucokinase has lower affinity for glucose than do the other hexokinases. HXK I has been shown to be expressed in brain, kidney and heart tissues as well as in hepatoma cell lines. HXK II is involved in the uptake and utilization of glucose by adipose and skeletal tissues. Of the hexo-kinases, HXK III has the highest affinity for glucose. Glucokinase is expressed in pancreatic beta cells where it functions as a glucose sensor, determining the "set point" for Insulin secretion.

REFERENCES

1. Katzen, H.M., et al. 1965. Multiple forms of hexokinase in the rat: tissue distribution, age dependency, and properties. Proc. Natl. Acad. Sci. USA 54: 1218-1225.
2. Arora, K.K., et al. 1990. Glucose phosphorylation in tumor cells. Cloning, sequencing, and overexpression in active form of a fulllength cDNA encoding a mitochondrial bindable form of hexokinase. J. Biol. Chem. 265: 6481-6488.
3. Stoeffel, M., et al. 1992. Human glucokinase gene: isolation, characterization, and identification of two missense mutations linked to early-onset non-Insulin-dependent (type 2) diabetes mellitus. Proc. Natl. Acad. Sci. USA 89: 7698-7702.
4. Deeb, S.S., et al. 1993. Human hexokinase II: sequence and homology to other hexokinases. Biochem. Biophys. Res. Commun. 197: 68-74.
5. Palma, F., et al. 1996. Purification and characterization of the carboxyl-domain of human hexokinase type III expressed as fusion protein. Mol. Cell. Biochem. 155: 23-29.

CHROMOSOMAL LOCATION

Genetic locus: HK1 (human) mapping to 10q22.1.

SOURCE

HXK I (4D7) is a mouse monoclonal antibody raised against full length recombinant HXK I of human origin.

PRODUCT

Each vial contains 100 µg IgG_{2a} in 1.0 ml PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

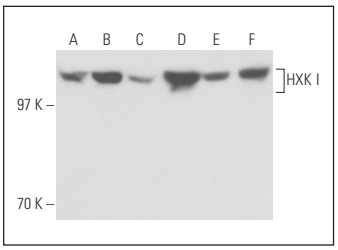
HXK I (4D7) is recommended for detection of HXK I of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for HXK I siRNA (h): sc-39044, HXK I shRNA Plasmid (h): sc-39044-SH and HXK I shRNA (h) Lentiviral Particles: sc-39044-V.

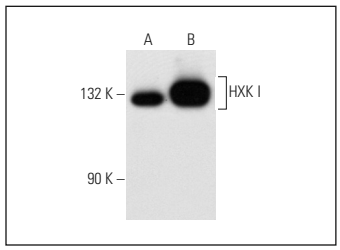
Molecular Weight of HXK I: 120 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, Hep G2 cell lysate: sc-2227 or HXK I (h2): 293T Lysate: sc-170521.

DATA



HXK I (4D7): sc-80978. Western blot analysis of HXK I expression in HeLa (**A**), Hep G2 (**B**), SK-N-MC (**C**), ME-180 (**D**), HEL 92.1.7 (**E**) and JAR (**F**) whole cell lysates.



HXK I (4D7): sc-80978. Western blot analysis of HXK I expression in non-transfected: sc-117752 (**A**) and human HXK I transfected: sc-170521 (**B**) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Koziel, A., et al. 2012. The influence of high glucose on the aerobic metabolism of endothelial EA.hy926 cells. Pflugers Arch. 464: 657-669.
2. Broniarek, I., et al. 2016. The effect of chronic exposure to high palmitic acid concentrations on the aerobic metabolism of human endothelial EA.hy926 cells. Pflugers Arch. 468: 1541-1554.
3. Koziel, A. and Jarmuszkiewicz, W. 2017. Hypoxia and aerobic metabolism adaptations of human endothelial cells. Pflugers Arch. 469: 815-827.
4. Broniarek, I., et al. 2020. The influence of statins on the aerobic metabolism of endothelial cells. Int. J. Mol. Sci. 21: 1485.
5. Sabbir, M.G., et al. 2021. Loss of β-Arrestins or six G_{α} proteins in HEK293 cells caused Warburg effect and prevented progesterone-induced rapid proteasomal degradation of progesterone receptor membrane component 1. J. Steroid Biochem. Mol. Biol. 214: 105995.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.